We claim:

1. A null mutant rodent comprising in its germ cells an artificially induced *PTTG* null mutation.

2. The null mutant rodent of claim 1, wherein functional PTTG protein is not expressed in the somatic cells of said rodent.

3. The null mutant rodent of claim 1, wherein the cells of said rodent lack the ability to endogenously express functional PTTG protein.

4. The null mutant rodent of claim 1, wherein both *PTTG* genes have been artificially mutated by way of homologous recombination.

5. The null mutant rodent of claim 1, wherein the *PTTG* null mutant was generated by a mating of a male rodent and female rodent of the same species each bearing at least one artificially mutated *PTTG* allele.

6. The null mutant rodent of claim 5, wherein said at least one mutated *PTTG* allele is generated by way of homologous recombination.

7. The null mutant rodent of claim 5, wherein said at least one mutated *PTTG* allele is generated by way of homologous recombination in an embryonic stem cell.

8. The null mutant rodent of claim 7, wherein the embryonic stem cell line is murine ES J-1.

9. The null mutant rodent of claim 7, wherein said embryonic stem cell is injected into a blastocyst, and wherein the blastocyst is implanted into a pseudopregnant rodent.

10. The null mutant rodent of claim 5, wherein said at least one mutated *PTTG* allele is generated by way of homologous recombination in an embryonic stem cell, and wherein at least one rodent genomic copy of the *PTTG* gene in the embryonic stem cell recombines with a targeting vector containing a selectable genetic marker sequence, such that said targeting vector is inserted into the genome of said embryonic stem cell.

11. The null mutant rodent of claim 5, wherein said at least one mutated *PTTG* allele contains a deletion of the translation start site.

12. The null mutant rodent of claim 5, wherein said at least one mutated *PTTG* allele contains a deletion of the KOZAK region.

13. The null mutant rodent of claim 5, wherein said at least one mutated *PTTG* allele contains a deletion of a segment of the endogenous *PTTG* gene promoter region.

14. The null mutant rodent of claim 5, wherein said at least one mutated *PTTG* allele contains a deletion of the transcription start codon.

15. The null mutant rodent of claim 5, wherein said at least one mutated *PTTG* allele is generated by way of site specific recombination.

16. The null mutant rodent of claim 5, wherein said at least one mutated *PTTG* allele is generated by way of transpositional recombination.

17. The null mutant rodent of claim 5, wherein said at least one mutated *PTTG* allele is generated by way of a frame shift mutation.

18. The null mutant rodent of claim 5, wherein said at least one mutated *PTTG* allele is generated by way of homologous recombination in a germ cell.

19. The null mutant rodent of claim 18, wherein the germ cell is an oocyte.

20. The null mutant rodent of claim 18, wherein the germ cell is a male germ cell.

21. A somatic cell obtained from the null mutant rodent of claim 1.

22. A cell line derived from the cell of claim 21.

23. A cell line derived from a cell obtained from the null mutant rodent of claim 1.

24. A germ cell obtained from the null mutant rodent of claim 1.

25. The null mutant rodent of claim 1, wherein the rodent is a mouse.

26. The null mutant rodent of claim 1, wherein the rodent is a rat.

27. A null mutant rodent comprising in its germ cells an artificially induced *PTTG* null mutation, wherein said mutation results in said rodent exhibiting at least one phenotype selected from the group consisting of hyperglycemia, hypoinsulinaemia, hypoleptinemia, diabetes, chromosomal aneuploidy, premature centromere division, chromosomal damage, aberrant mitotic cellular division, thrombocytopenia, thymic hyperplasia, splenic hypoplasia, testicular hypoplasia, and female subfertility, the prevalence of which is greater than in a rodent lacking said mutation.

28. A rodent whose germ line comprises an artificially induced *PTTG* null mutation, wherein both mutated *PTTG* genes are transmitted to said rodent by a mating of a male rodent and female rodent of the same species each bearing at least one artificially mutated *PTTG* allele; said at least one mutated *PTTG* allele is generated by way of homologous recombination with a targeting vector; and said targeting vector further comprises a selectable genetic marker; said targeting vector contains a polynucleotide sequence comprising a segment of *PTTG* genomic DNA or a *PTTG* cDNA spanning the *PTTG* KOZAK sequence from which the KOZAK sequence has been deleted and replaced with polynucleotides exogenous to the *PTTG* gene, and said exogenous polynucleotides are flanked by at least about 200 polynucleotide base pairs that are complementary to polynucleotide regions of an endogenous *PTTG* gene which flank the endogenous KOZAK sequence.

29. A rodent whose germ line comprises an artificially induced *PTTG* null mutation, wherein both mutated *PTTG* genes are transmitted to said rodent by a mating of a male rodent and female rodent of the same species each bearing at least one artificially mutated *PTTG* allele; said at least one mutated *PTTG* allele is generated by way of homologous recombination with a targeting vector; and said targeting vector further comprises a selectable genetic marker; said targeting vector contains a polynucleotide sequence comprising a segment of *PTTG* genomic DNA or a *PTTG* cDNA spanning the PTTG translation start site from which the translation start site has been deleted and replaced with polynucleotides exogenous to the *PTTG* gene; and said exogenous polynucleotides are flanked by at least about 200 polynucleotide base pairs that are complementary to polynucleotide regions of an endogenous *PTTG* gene which flank the endogenous translation start site.

30. A rodent whose germ line comprises an artificially induced *PTTG* null mutation, wherein both mutated *PTTG* genes are transmitted to said rodent by a mating of a male rodent and female rodent of the same species each bearing at least one artificially mutated *PTTG* allele; said at least one mutated *PTTG* allele is generated by way of homologous recombination with a targeting vector; and said targeting vector further comprises a selectable genetic marker; said targeting vector contains a polynucleotide sequence comprising a segment of *PTTG* genomic DNA or a *PTTG* cDNA spanning the *PTTG* transcription start codon from which the transcription start site has been deleted and replaced with polynucleotides exogenous to the *PTTG* gene; and said exogenous polynucleotides are flanked by at least about 200 polynucleotide base pairs that are complementary to polynucleotide regions of an endogenous *PTTG* gene which flank the endogenous transcription start codon.

31. Use of the null mutant rodent of any of claims 1, 27-30 in the study of mammalian physiology at the cellular, tissue, and/or organismal level.

32. Use of the null mutant rodent of any of claims 1, 27-30 in the study of mammalian physiology at the cellular, tissue, and/or organismal level, wherein the physiological role of PTTG is examined in connection with regulation of a physiological phenomenon selected from the group consisting of diabetes, hyperglycemia, hypoinsulinaemia, hypoleptinemia.

33. Use of the null mutant rodent of any of claims 1, 27-30 in the study of mammalian physiology at the cellular, tissue, and/or organismal level, wherein the physiological role of PTTG is examined in connection with regulation of a physiological phenomenon selected from the group consisting of chromosomal aneuploidy, premature centromere division, chromosomal damage, the mitotic cellular pathway, and cell cycle control.

34. Use of the null mutant rodent of any of claims 1, 27-30 in the study of mammalian physiology at the cellular, tissue, and/or organismal level, wherein the physiological role of PTTG is examined in connection with regulation of a physiological phenomenon selected from the group consisting of thrombocytopenia, thymic hyperplasia, and splenic hypoplasia.

35. Use of the null mutant rodent of any of claims 1, 27-30 in the study of mammalian physiology at the cellular, tissue, and/or organismal level, wherein the physiological role of PTTG is examined in connection with regulation of a physiological phenomenon selected from the group consisting of testicular hypoplasia and female subfertility.

36. An animal model for diabetes comprising the null mutant rodent of any of claims 1, 27-30.